What is claimed is:

1. A method of inhibiting growth of cancer cells in a patient, comprising:
administering to said patient an effective amount of an antagonist of STAT (signal transducer and activator of transcription) signaling, wherein said antagonist is an inhibitor of STAT dimerization, an inhibitor of a tyrosine kinase capable of phosphorylating STAT, an antagonist of SH2-pY interactions, a dominant negative STAT protein, an antagonist of STAT DNA binding, a tyrphostin inhibitor, an antagonist of STAT-dependent gene transactivation, an antagonist of IL-6 receptor activation, an antagonist of a cytokine that constitutively activates STAT, an antagonist of a growth factor that constitutively activates STAT, or mixtures thereof in a pharmaceutically acceptable carrier.

2. The method of claim 1, wherein said STAT is STAT3.

3. The method of claim 1, wherein said tyrosine kinase is Jak, Src, or BCR-Abl.

4. A method of inducing apoptosis in cancerous cells of a patient, comprising:
administering to said patient an effective amount of an antagonist of STAT3 activation, in a pharmaceutically acceptable carrier.

5. A method of inhibiting tumorigenesis in a patient, comprising:
administering to said patient an effective amount of an antagonist of STAT3 activation, in a pharmaceutically acceptable carrier.

6. A method of inhibiting neoplastic transformation of a cell of a subject, comprising:
administering to said patient an effective amount of an antagonist of STAT3 activation, in a pharmaceutically acceptable carrier.

7. A method of increasing the efficiency of a chemotherapeutic agent for selectively killing cancer cells in a patient, comprising:
administering to said patient an effective amount of an antagonist of STAT3 activation, in a pharmaceutically acceptable carrier; and

further administering to said patient at least one dose of said chemotherapeutic agent.

8. A method of increasing the efficiency of radiation therapy for selectively killing cancer cells in a patient, comprising:

administering to said patient an effective amount of an antagonist of STAT3 activation, in a pharmaceutically acceptable carrier; and

further administering to said patient at least one application of said radiation therapy.

9. A method to induce apoptosis in the cells of a solid tumor, comprising:

contacting said tumor with a solution containing an effective quantity of an antagonist of STAT signaling, wherein said antagonist is an inhibitor of STAT dimerization, an inhibitor of STAT tyrosine phosphorylation, an antagonist of SH2-pY binding, an antagonist of STAT DNA binding, a tyrphostin inhibitor, an antagonist of transactivation of a gene by STAT, an antagonist of IL-6 receptor activation, an antagonist of a cytokine that constitutively activates STAT, an antagonist of a growth factor that constitutively activates STAT, or mixtures thereof in a pharmaceutically acceptable carrier; and

electroporating said tumor, whereby said antagonist of STAT signaling enters said cells of said tumor,

whereby apoptosis is induced in said cells.

10. A method to induce apoptosis in the cells of a solid tumor, comprising:

contacting said tumor with a solution containing an effective quantity of a DNA construct encoding a dominant-negative variant STAT3 protein and further encoding genetic control elements directing expression of said protein within said cells of said tumor; and

applying electroporating means to said tumor, whereby said DNA is placed within said cells of said tumor,

whereby apoptosis is induced in said cells.

11. The method as in claim 10, wherein said dominant-negative variant STAT3 protein is STAT3b.

12. A method of treating a cancer of a patient, wherein said cancer has constitutively activated JAK-STAT signaling, comprising:

administering to said patient an effective amount of a tyrphostin inhibitor in a pharmaceutically acceptable carrier; and

further administering to said patient an effective amount of IL-12 in a pharmaceutically acceptable carrier.

13. The method as in claim 12, wherein said tyrphostin inhibitor is AG490.

14. The method as in claim 12, wherein said tyrphostin inhibitor is AG17, AG213 (RG50864), AG18, AG82, AG494, AG825, AG879, AG1112, AG1296, AG1478, AG126, RG13022, RG14620, AG555, or related compounds.

15. A cell line for screening compounds for specific inhibition of STAT signaling, comprising:

a viable cell, said cell comprising:

a first reporter gene operably linked to a STAT3-responsive promoter, and

a second reporter gene operably linked to a promoter that is STAT-3 independent.

16. The cell line of claim 16, wherein said first and second reporter genes encode luciferase protein variants with different light emission spectra.

17. The cell line of claim 16, wherein said cell is a eucaryote.

18. A method for high throughput screening of compounds that specifically inhibit STAT signaling in the cells of claim 17, said method comprising:

contacting a multi-well receptacle with a suspension of said cells in a suitable liquid medium, such that each well of said receptable contains a separate sample of said cells,

contacting said cells with at least one of said compounds,

measuring light spectra emitted by said cells, and

analyzing said light spectra to determine relative activation of said first and second reporter genes,

whereby compounds specifically inhibiting activation of said STAT3-responsive promoter are identified.

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